

Proceedings of the British Cardiac Society

THE 58TH ANNUAL GENERAL MEETING of the British Cardiac Society was held at the Medical and Biological Sciences Building of the University of Southampton on Wednesday and Thursday, 4 and 5 April 1979. The President, W. SOMERVILLE, took the Chair during private business. At the scientific sessions the Chair was taken by A. M. JOHNSON.

Abstracts of papers

Coronary artery disease in the transplanted heart: failure to develop abnormal lactate metabolism on atrial pacing

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Accelerated coronary artery disease is a frequent problem in the transplanted heart. As the patient with a denervated heart cannot experience angina, it is difficult to judge the clinical significance of advanced coronary artery disease. Abnormal lactate metabolism after atrial pacing has been shown reliably to reflect 2 or 3 vessel coronary artery disease. We therefore performed atrial pacing and coronary sinus lactate studies in 6 cardiac transplant recipients in an attempt to determine objectively the presence or absence of myocardial ischaemia. Coronary angiography was performed in all 6 patients, 3 of whom had advanced coronary artery disease and 3 normal coronary arteries. Pacing was at 160 beats/minute for at least 10 minutes with 12-lead electrocardiograph monitoring of the ST segment. All studies were without complications and symptoms.

All 6 patients had normal lactate metabolism on peak pacing as judged by the absence of lactate production and an extraction ratio greater than 10 per cent. Of the 3 patients with coronary artery disease, 2 subsequently underwent retransplantation, 1 for cardiac failure and the other for massive myocardial infarction.

The transplanted heart with advanced coronary artery disease appears unable to develop abnormal lactate metabolism on atrial pacing. The mechanism is unclear, but may reflect loss of sympathetic innervation.

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Ambulatory ST segment monitoring: problems, pitfalls, and solutions

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Ambulatory tape recording systems which are suitable for detection of arrhythmias are not adequate for accurate detection and quantification of ST segment shift. Low frequency artefact and baseline drift are major problems. Systematic investigation has resulted in recommendations for skin preparation and a new electrode design which minimises connection artefacts. Three tape recorders used for signal storage have been evaluated: the Medilog Mark I, the Cardiodyne real-time sampling recorder, and the Medilog Mark II. High fidelity electrocardiographs were recorded during and after exercise in 20 patients with ischaemic heart disease using the Marquette CASE system and the 3 recorders simultaneously. The taped recordings were compared with the CASE recording as a standard. Medilog I showed distorted calibration signals, unpredictable ST depression, and slope distortion and baseline drift. Medilog II had good calibration with a stable baseline and excellent ST reproducibility. The best reproduction was obtained with the Cardiodyne. Similar results were obtained from 24-hour ambulatory outpatient records in the same patients. Accurate and repeatable ST segment deviations can only be obtained by using an electrode system which gives a stable signal and a recorder whose physical characteristics are adequate. This new electrode system will give reliable records with Medilog II (24 hours) and the Cardiodyne (28 s in 15 min) but not with the Medilog I.

Effects of propranolol on left ventricular wall movement in patients with ischaemic heart disease

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In order to assess the effects of propranolol on left ventricular wall movement in ischaemic heart

disease, simultaneous echo-, phono-, and apex cardiograms were recorded and digitised in 16 patients before and after 0.1 mg/kg iv, or therapeutic oral dose for 1 week. In 9 (group A) contraction was synchronous, and in 7 (group B) there was evidence of incoordinate wall movement. There were small reductions in heart rate, peak VCF, and extent of dimension change ($P < 0.05$ for each). In group A, the main abnormality was a 80 ms reduction in the duration of inward wall movement so that minimum dimension occurred prematurely with respect to mitral valve opening. Diastolic events were otherwise unaltered. In group B, apex-dimension relations and timing of A2 and mitral valve opening were abnormal under control conditions, and minimum dimension already occurred prematurely. Propranolol did not alter systolic events further, but aggravated diastolic abnormalities with further delay in mitral valve opening ($P < 0.001$), 'O' point ($P < 0.01$), prolongation of isovolumic relaxation ($P < 0.01$), and reduced rate of dimension increase ($P < 0.05$).

In ischaemic heart disease, therefore, the effect of propranolol administration is significantly modified by the presence of incoordinate contraction in a manner not predictable from the results in normal subjects.

Abnormal aortic valve echoes in mitral prolapse

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Abnormal aortic valve echograms were recorded in 6 patients with echocardiographic and clinical evidence of mitral prolapse.

No patient was considered to have Marfan's syndrome: 2 had clinical signs of mild aortic regurgitation but in the remaining patients, including 1 who subsequently had a myxomatous aortic valve replaced, aortic valve disease was not suspected clinically.

On cross-sectional echocardiograms, the aortic valve cusps appeared thickened but mobile. Three cusps were defined in each of the 4 valves imaged in transverse section.

M-mode scans showed normal motion of the

aortic cusps in systole but during diastole multiple echoes were recorded from the cusps in the centre of the aortic root. There was evidence of aortic root dilatation in 2 patients.

It is suggested that the echocardiographic features described reflect myxomatous degeneration of the aortic valve, and it is recommended that the investigation of patients with mitral prolapse should include echocardiographic assessment of the aortic valve.

Plasma noradrenaline concentrations during isometric and dynamic exercise in essential hypertension

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Plasma noradrenaline concentrations were studied in 15 essential hypertensives, before, during, and after isometric and dynamic exercise. Blood pressures were measured intra-arterially from the brachial artery. Isometric exercise comprised maximum grip strength for 30 seconds followed by 30 per cent of maximum for up to 4 minutes 30 seconds. The same subjects later performed dynamic exercise on a bicycle ergometer.

Resting arterial pressure, heart rate, and plasma noradrenaline were similar before each period of exercise. Mean arterial pressure increased by 29.1 to 144.6 ± 23.6 mmHg during isometric exercise ($P < 0.001$) and to 150 ± 25.4 mmHg during bicycling ($P < 0.001$).

Heart rate increased by 23 to 96.6 ± 13.5 bpm during isometric exercise ($P < 0.001$) and to 126.7 ± 16.3 bpm during bicycling ($P < 0.001$). Plasma noradrenaline concentration did not alter significantly during isometric exercise, increasing from 3704.4 ± 1196.8 pmol/l (626.8 ± 202.5 pg/ml) to 3949.1 ± 1341 pmol/l (668.2 ± 226.9 pg/ml), but rose from 3198.5 ± 843.4 pmol/l (541.2 ± 142.7 pg/ml) to 7705.6 ± 3212.1 pmol/l (1303.8 ± 543.5 pg/ml) during dynamic exercise ($P < 0.001$).

Noradrenaline increased more during dynamic exercise for the same mean pressure rise (heart rate was higher however). Previous observations of raised plasma noradrenaline during isometric exercise in patients with disturbed left ventricular function do not seem to apply to hypertensive subjects not in failure.

Computerised dichromatic earpiece densitometer for measurement of cardiac output

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Technical problems have prevented the routine use of earpiece densitometry to measure cardiac output. We have evaluated a precalibrated, computerised, dichromatic earpiece densitometer by comparison of cardiac output with that simultaneously determined using a cuvette densitometer. Correction for ear blood content and background dye for the individual patient used a 'bloodless ear' technique.

Thirty-four patients were studied during diagnostic cardiac catheterisation. Cardiac output was determined after duplicate injections of indocyanine green in the main pulmonary artery and in the femoral vein. In 4 patients with low cardiac outputs the dilution curves were unacceptable. For the remaining 30 patients measured cardiac output was in the range 2.20 to 7.15 l/min. Comparison of computer and manual processing of the dye dilution curves showed excellent agreement (r 0.994; SD 1.47%). Reproducibility of cardiac output measurements by earpiece densitometry (SD 5.2%) was comparable with that using the cuvette (SD 5.3%). Comparison of simultaneously determined cardiac output using both instruments showed good correlation (r 0.83; SD 7.2%) after pulmonary arterial injection of dye and (r 0.78; SD 8.3%) after femoral venous injection.

The instrument offers a useful technique for the non-invasive measurement of cardiac output at rest. Further applications are being studied.

Relation between symptoms and arrhythmias in patients with sick sinus syndrome

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The relation between symptoms and documented arrhythmias has been studied in 94 patients with sick sinus syndrome (mean age 67.6 years). 22 patients were being treated medically, 32 had implanted cardiac pacemakers, and the remaining 40 were assessed both before and after pacemaker implantation.

Documented arrhythmias were grouped into 5 categories: (a) major atrial arrhythmias; (b) major ventricular arrhythmias; (c) profound bradycardia;

(d) minor atrial or ventricular arrhythmias including minor bradycardia; (e) normal sinus rhythm.

Of the patients treated medically, 76 per cent experienced major atrial arrhythmias and 97 per cent of these caused symptoms, 32 per cent of these patients had major ventricular arrhythmias apart from bradycardia; 82 per cent had major bradycardia and the great majority of both fast and slow ventricular arrhythmias correlated with symptoms. Only 29 per cent of the minor arrhythmias were associated with symptoms.

Of this medical group, 19 per cent complained of symptoms despite 24-hour electrocardiograms showing only stable sinus rhythm. The incidence of arrhythmias in patients with pacemakers was very much lower for both major atrial arrhythmias (33%) and major ventricular arrhythmias (12.8%). When such arrhythmias did occur about two-thirds of the patients had associated symptoms.

In the third group of patients implantation of a pacemaker reduced the incidence of symptoms to approximately one-third of the previous level and this occurred for all types of arrhythmia. It was apparent that arrhythmias in all categories a-d diminished spontaneously during sleep.

We conclude that there is only fair correlation between the patients' symptoms and the documented arrhythmias; the correlation is best for profound bradycardia and rapid atrial arrhythmias. Implantation of a cardiac pacemaker considerably reduces the subjective complaints associated with both fast and slow arrhythmias.

Prognosis in sinoatrial disorder (sick sinus syndrome)

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The 5-year survival rate in patients with sinoatrial disorder has been compared with that of the general population. The relative risk of mortality has been assessed according to the severity of the disease and whether or not a pacemaker was implanted.

Over a period of 10 years 386 patients were collected from the Devon Heart Block Survey. All had a chronic sinus bradycardia and were classed as either group 1 if there were additional major electrocardiographic changes (N = 160) or group 2 if there was uncomplicated sinus bradycardia (N = 226).

The mortality in the 2 groups was similar in the 5 years after entering the survey. In addition there

was no significant difference in the relative risk of mortality as compared with a general population matched for age and sex, that is,
 $\frac{\text{Proportion dead in sample}}{\text{Proportion dead in population}} = 0.39, 1.04, 0.87,$
 over the first 5 years. Survival of the 61 paced patients was no different from that of the unpaced patients in either group.

It is concluded that survival of these patients with chronic sinuatrial disorder is similar to that of the general population and does not appear to be materially altered by pacemaker implantation.

Study to determine normal fetal heart rate and rhythm

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The fetal heart was recorded for 5 minutes onto cassette from 963 randomly selected mothers between 36 weeks gestation and term. The recordings were printed as phonocardiograms and analysed directly by listening to rhythm and measuring rate. Twelve fetuses (1%) had premature beats ranging from 3 to 102 per 5 minute recording. Episodes of bradycardia, rate 70 to 110/minute ranging from 3 to 25 seconds duration, occurred in 18 (1.9%). Episodes of bradycardia (± 70 /minute) ranging from 5 seconds to 5 minutes duration, occurred in 7 (0.7%). Episodes of tachycardia (> 180 /minute) ranging from 30 to 90 seconds in duration occurred in 5 (0.5%). Recordings were replayed through a standard antepartum fetal heart rate monitor. All of the premature beats, 11 of the 18 fetal heart rates between 70 and 110/minute, 5 of the 7 rates below 70/minute, and 2 of the 5 rates over 180/minute were not detected. These failures of standard monitors to detect the majority of rhythm and rate changes in the fetus are the result of an inbuilt electronic circuit evolved to avoid artefact. This lost information could be relevant to hitherto unexplained antepartum asphyxia and intrauterine death.

Study of heart rate and rhythm in normal schoolchildren

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Cardiac arrhythmias associated with sinus node dysfunction have been suggested as a cause of fits, faints, and funny turns in childhood. However, information on normal heart rate and rhythm is poorly documented and therefore single 24-hour electrocardiographic recordings were performed on 90 randomly selected healthy 7 to 11-year-old children. The mean highest heart rate over 9 beats was 164 ± 16 , the mean lowest heart rates were 49 ± 6 over 3 beats, and 56 ± 6 over 9 beats duration. At their lowest heart rates, 42 (46%) had intermittent junctional rhythms. One child had episodes of complete 2:1 Wenckebach atrioventricular nodal block and 7 had intermittent prolongation of the PR interval (> 0.20 s). Nineteen had isolated atrial premature beats. In a randomly selected subgroup of 29 children, 23 (79%) had sinatrial Wenckebach block and 5 (17%) had 2:1 sinatrial block patterns.

A history and clinical examination were taken on all children studied and these showed no correlation between variations in rhythm and rate and clinical findings. This study shows that normal children have variations in heart rate and rhythm hitherto considered to be abnormal and, therefore, must be carefully considered before making a diagnosis of conducting system disorders such as the sick sinus syndrome.

Comparison between results of primary repair of large ventricular septal defects before and after the age of 1 year

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Forty-five patients with large ventricular septal defects and a pulmonary artery pressure in excess of 0.75 of systemic pressure have undergone primary closure of their defect. Twenty-nine patients were under the age of 1 year (group A) and 16 patients were over 1 year (group B). Pre-operatively the pulmonary vascular resistance was 1.5 to 6 units in group A and 2 to 8.5 units in group B. There were 2 early deaths in group A and no late deaths in either group. Follow-up was from 9 to 92 months (mean 40 months). All children are asymptomatic, though 1 child in group B is on cardiac drugs. Eighteen patients have been investigated 1 to 4 years after operation in group A and 9 patients, 9 months to 7 years after operation in group B. There were no residual shunts in either group. The peak pulmonary artery pressure was

less than 25 mmHg at rest and on exercise in all 18 patients in group A, while in group B only 1 patient had a normal pulmonary artery pressure at rest while in 8 patients the pulmonary artery/systemic ratio was 0.33 and 0.6, even at rest. The pulmonary vascular resistance was 2 units, or less, in all patients in group A and 1.5 to 6 units in group B. Right ventricular end-diastolic pressure at rest was below 10 mmHg in both groups. Left ventricular ejection fraction was between 65 to 85 per cent in group A and 55 to 75 per cent in group B.

It is concluded that the late results of primary correction of large ventricular septal defects in infancy are significantly better than those in older children.

Total correction of tetralogy of Fallot in adult life—a worthwhile operation?

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Between October 1969 and September 1978, 18 patients between the ages of 16 and 50 years (mean 25.5 years) underwent total correction of tetralogy of Fallot. All patients were operated upon because of recent deterioration in symptoms. Seven patients had had previous palliative operations in childhood and 2 had had attempts at total correction in other centres. Severe tricuspid regurgitation was present in 1 and triple coronary artery disease in 1. Three patients had reconstruction of the right ventricular outflow tract with a single cusp of a fresh aortic homograft. Additional triple aortocoronary vein grafts were performed in 1 and resection of a left ventricular diverticulum in 1. There were no early or late deaths. The patients have been followed up for between 4 and 111 months (mean 43 months) and have had considerable symptomatic improvement. Five patients have been restudied 1 to 3 months after operation and all have LV/RV ratios of less than 0.45 with evidence of improved left ventricular function.

It is concluded that total correction of tetralogy of Fallot in adult life can be carried out safely and can lead to considerable symptomatic improvement.

Underdeveloped right ventricle with right-to-left shunting interatrial defect: recognition and results of surgery

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Studies were made on 7 patients, 3 to 19 years of age, with cyanosis caused by right-to-left shunts at atrial level and hypoplastic abnormalities of the right ventricle. Common clinical features were cyanosis, clubbing, giant 'a' wave, quiet parasternal area, soft systolic murmur, inspiratory third and/or atrial sound, close second sound, right atrial prominence on electrocardiogram, and radiograph with pulmonary oligemia. Cyanosis was progressive. Investigation confirmed shunting with arterial oxygen saturations of 82 to 95 per cent at rest, falling to 80 to 91 per cent with effort, raised right ventricular end-diastolic pressures, and tricuspid valve gradients in 4. Right ventricular angiographic appearances were bizarre, with reduced ejection, small tricuspid ring, smooth inflow, and multiple pouches. The shunt was closed in 4, in 1 the dominant lesion proved to be an abnormal adherent Eustachian valve above the small tricuspid ring. Early postoperative problems included right heart failure in all and arrhythmias in 2. All are well 6 months to 3 years later.

Of the 7 patients, 5 had family histories of congenital abnormalities. Three presented with an incorrect diagnosis of inoperable Eisenmenger. Diagnostic accuracy is essential since surgery may abolish cyanosis, and the long-term outlook may be good if operation is carried out early enough to allow the right ventricle to develop normally and prevent irreversible hypoplasia.

Mechanism of sudden death in postoperative tetralogy of Fallot

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Late sudden death occurs after the correction of tetralogy of Fallot in 1 to 3 per cent of patients. The mechanism is controversial: conduction disturbances progressing to complete heart block or serious ventricular arrhythmia have been suggested. We reviewed the pre-, perioperative, and follow-up electrocardiograms of 176 patients after correction of tetralogy of Fallot age 3-16, mean 8 years, for evidence of conduction disturbances and arrhythmia. 24-hour ambulatory electrocardiographic monitoring was performed in 65 patients.

Perioperative electrocardiograms showed complete right bundle-branch block in 83 per cent, and

right bundle-branch block and left anterior hemiblock in 9 per cent, trifascicular block in 3 per cent, and mean frontal QRS axis shift greater than 90 degrees in 16 per cent. Seven patients died perioperatively: 2 had complete heart block and 2 had frequent ventricular extrasystoles. Transient perioperative arrhythmias were: complete heart block in 8 patients, frequent ventricular extrasystoles in 14 patients, ventricular fibrillation in 1 patient.

Follow-up was 1 to 20 years (mean 12 years). There were 5 late sudden deaths; none of these patients had documented perioperative arrhythmias or conduction disturbances. During follow-up, the electrocardiogram showed a progressive axis shift with development of left anterior hemiblock in 12 per cent of patients, 2 of the sudden deaths occurring in this group. 24-hour ambulatory electrocardiograms showed 4 patients (6%) with episodes of supraventricular tachycardia, 19 patients (30%) with frequent multiform or coupled ventricular extrasystoles, 8 patients (12%) with intermittent first degree heart block, 4 patients with atrioventricular dissociation, and 10 patients (15%) with sinus pauses. The high frequency of ventricular arrhythmia was not confined to the early postoperative or late left anterior hemiblock groups.

We have shown a high incidence of late electrocardiographic axis shift with development of left anterior hemiblock associated with sudden death. This may represent progressive conduction disease, and this group may be at particular risk from ventricular arrhythmia and complete heart block.

Amiodarone in management of chronic refractory ventricular tachycardia

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Previous reports have shown the efficacy of the drug amiodarone in the management of atrial arrhythmias especially atrial fibrillation associated with the Wolff-Parkinson-White syndrome: it has been less effective against reciprocating atrioventricular tachycardias associated with the Wolff-Parkinson-White syndrome and paroxysmal atrioventricular node re-entry. We have studied its effects in 10 patients (aged 24 to 61 years) with recurrent ventricular tachycardia refractory to all conventional drug treatment (duration of symptoms 6 to 48 months). Seven patients had required repeated electroversions. Eight patients were studied electrophysiologically using programmed stimulation before amiodarone (5 re-entry and 3 focal mecha-

nism). Ambulatory electrocardiographic monitoring was performed both before and serially during subsequent treatment with amiodarone. (Follow-up 4 to 16 months, mean 10 months.)

Six patients (congestive cardiomyopathy, 3: ventricular aneurysm, 2: idiopathic ventricular tachycardia, 1) were asymptomatic on amiodarone with no ventricular tachycardia on ambulatory electrocardiographic monitoring. There was also pronounced reduction in the number of ventricular premature beats. The average maintenance dose was 200 mg per day. Three patients (old infarct, 1; mitral valve prolapse, 2) were improved with only either short runs of ventricular tachycardia or frequent premature beats on ambulatory electrocardiographic monitoring. Maintenance dosage was between 300 and 600 mg per day for these patients. One patient with congestive cardiomyopathy and re-entrant left ventricular tachycardia showed no improvement on 600 mg of amiodarone per day. One patient had ocular side-effects of treatment which settled on reducing the dose without loss of antiarrhythmic effect.

We conclude that amiodarone has an important place in the management of chronic refractory ventricular tachycardia.

Treatment of resistant non-ischaemic ventricular tachycardia

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When ventricular tachycardia fails to be controlled by one of the wide variety of drugs available, the disabling symptoms and inherent dangers require that alternative methods of treatment be sought. We describe 2 patients with ventricular tachycardia not caused by cardiac ischaemia in whom surgery was required. The surface electrocardiogram of a patient with mitral valve prolapse suggested that the tachycardia arose from a region of the ventricle closely related to the mitral valve; haemodynamically unimportant mitral regurgitation in sinus rhythm assumed major haemodynamic importance during tachycardia. Epicardial mapping at operation showed the tachycardia to be closely related to the posterior papillary muscle; after mitral valve replacement the patient is free of arrhythmias. In a second case the development of a ventricular aneurysm after non-penetrating cardiac trauma was associated several years later with frequent incapa-

citating episodes of ventricular tachycardia. Cryo-ablation after epicardial mapping has completely abolished these episodes. The follow-up in both these cases now exceeds 18 months.

These 2 patients illustrate the importance of accurately identifying the underlying cause of any resistant ventricular tachycardia. Epicardial mapping with surgical ablation may be the only means of control of such an arrhythmia in patients who fail to respond to drug treatment.

Relation between sites of ventricular tachycardia and underlying ventricular disease: implications for therapy

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The relation between the site of ventricular tachycardia, and of any underlying ventricular disease was examined in 38 patients with chronic recurrent ventricular tachycardia. The arrhythmia was localised in each case from its electrocardiographic appearance in all 12 conventional surface leads. Transvenous intracardiac mapping was also used in 28, as was direct mapping during cardiac surgery in 11. Ventricular disease was localised angiographically and sometimes also by nuclear and echocardiography. In 21 patients with coronary artery disease, chronic recurrent ventricular tachycardia arose from sites of prior myocardial infarction in 20. Fourteen of these had a ventricular aneurysm. The remaining patient with high septal chronic recurrent ventricular tachycardia had non-corresponding myocardial infarction but critical stenosing coronary artery disease in the 2 vessels supplying the apparently undamaged septum. Bypass grafting failed to abolish the chronic recurrent ventricular tachycardia.

While a causal relation was probable in 7 with congestive cardiomyopathy, the site of chronic recurrent ventricular tachycardia was very variable and was not associated with localised disease. In 4 patients with mitral valve prolapse, chronic recurrent ventricular tachycardia was localised to, but not specific for, this abnormality. In 2 patients with surgically corrected tetralogy of Fallot, chronic recurrent ventricular tachycardia arose from the region of the pulmonary outflow tract in 1, and from the lower anterior septum in the other. One patient with both idiopathic left ventricular aneurysm and aortic valve disease had chronic recurrent ventricular tachycardia not associated with either. The focus was successfully localised and ablated after both aortic valve surgery and aneurys-

mectomy had failed. Three had idiopathic chronic recurrent ventricular tachycardia all arising from the right ventricular anterior free wall.

We conclude that the localisation of ventricular tachycardia using 12 lead surface electrography is both valid and useful in the management of such arrhythmias.

Response of paroxysmal atrial flutter to intravenous disopyramide phosphate and overdrive atrial pacing

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Ten patients with spontaneous paroxysmal atrial flutter were investigated electrophysiologically. Three had Wolff-Parkinson-White syndrome, 3 Lown-Ganong-Levine syndrome, and 1 had concealed Wolff-Parkinson-White. Paroxysmal atrial flutter was initiated by right atrial pacing and electrograms from the right atrium and coronary sinus were observed for 5 minutes to ensure stable biatrial flutter.

Paroxysmal atrial flutter was terminated by 10 second bursts of right atrial pacing at rates 10 to 100 bpm faster than its intrinsic rate in only 2 patients. Paroxysmal atrial flutter (reinitiated in 2 patients) was then treated with intravenous disopyramide phosphate, 2 mg/kg body weight, infused over 5 minutes. In all 10 patients the atrial rate slowed from 320 ± 43 bpm to 217 ± 27 bpm ($P < 0.001$) and paroxysmal atrial flutter terminated in 1. Though the mean ventricular rate fell from 158 ± 54 bpm to 144 ± 47 bpm (NS) atrioventricular conduction block decreased and left 2 patients with higher ventricular rates. In 9 patients right atrial pacing, repeated after disopyramide phosphate, terminated 7 arrhythmias. In 1 patient right atrial pacing was successful before but not after disopyramide phosphate.

In this study right atrial pacing and disopyramide phosphate, singly and in combination, terminated paroxysmal atrial flutter in 9 of the 10 patients and it is suggested that this combination reduces the need to proceed to DC cardioversion.

Comparison of atrial arrhythmias with atrial stimulation threshold and conduction times in patients with sick sinus syndrome

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Thirty-two patients have been investigated by repeated 24-hour electrocardiographic monitoring and also by measurements of atrial and ventricular stimulation thresholds. Intra-atrial conduction time was also measured in 2 ways: (1) as the maximum P wave duration recorded on multiple lead surface electrocardiograms with a rapid paper speed, (2) as delay between recordings from the high right atrium (near the sinus node) and the low right atrium (near the tricuspid ring).

Patients with atrial enlargement detected either by electrocardiogram or by echocardiogram were excluded. Of the 32 patients, 10 were normal controls, 10 had complete heart block, and 12 had sick sinus syndrome.

Of the patients with sick sinus syndrome 83.3 per cent had atrial arrhythmias as opposed to 30 per cent of patients with complete heart block and 10 per cent of patients with normal sinus rhythm.

Intra-atrial conduction time was significantly longer in patients with sick sinus syndrome than in those with complete heart block or normal sinus rhythm ($P < 0.001$).

The stimulation threshold in the atrium, with a 1 ms impulse and a C51 electrode was 2.2V for patients with sick sinus syndrome, 1.25V for patients with complete heart block, and 0.9V for normal subjects. The ventricular stimulation thresholds in the same groups were 0.62V, 0.55V, and 0.58V, respectively. All values are means of the 3 lowest readings obtained.

There was a close correlation between patients with sick sinus syndrome who had frequent atrial arrhythmias, prolonged intra-atrial conduction times, and high atrial stimulation thresholds. These 3 findings are regarded as indicating the extent of atrial disease, and, though the incidence of such abnormalities was much lower in the patients with complete heart block, this group also had evidence of atrial involvement. The information is of clinical significance when physiological pacing is being considered.

Electrophysiological properties of nifedipine in comparison with those of verapamil

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We have investigated the electrophysiological properties of nifedipine, a dihydropyridine derivative which has potent effects on the slow inward (calcium-dependent) channels of smooth and cardiac muscle and is an effective anti-anginal

agent. Twelve patients underwent electrophysiological testing to determine the nature of their arrhythmias. Seven were shown to have paroxysmal reciprocating atrioventricular tachycardia associated with the Wolff-Parkinson-White syndrome. Four patients had either paroxysmal atrial fibrillation or flutter and 1 had sinus node disease. Nifedipine ($7.5 \mu\text{g/kg}$ body weight) was administered over 3 minutes during paroxysmal reciprocating atrioventricular tachycardia or during rapid atrial pacing with 1:1 atrioventricular conduction. In no instance was paroxysmal reciprocating atrioventricular tachycardia terminated nor did 2nd degree atrioventricular block occur in those patients who were being paced.

Electrophysiological indices were measured again 5, 10, and 20 minutes after administration. The only significant change was an increase in the sinus rate presumably because of altered autonomic balance resulting from the negative inotropic action. No change was observed in the functional or effective refractory periods of the atrioventricular node; in contrast, verapamil (0.15 mg/kg) subsequently given to all patients consistently terminated paroxysmal reciprocating atrioventricular tachycardia and lengthened atrioventricular nodal refractoriness (functional or effective refractory periods).

Nifedipine is a potent 'calcium-antagonist' but does not possess the antiarrhythmic actions of some similar agents. While it appears safe to combine nifedipine with beta-blockers, these results raise the possibility that there may be more than one type of slow inward (calcium-dependent) channel in cardiac cell membranes.

Effect of variation in coronary artery anatomy on distribution of stenotic lesions

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The influence of 3 aspects of coronary artery anatomy (length of left main coronary artery, angle of bifurcation into anterior descending and circumflex branches, and artery of dominance) was investigated by review of 154 consecutive coronary angiograms. Ninety-five had critical stenoses ($\geq 50\%$ luminal diameter).

The mean left main coronary artery length in diseased arteries (10.6 mm) was shorter ($P < 0.50$) than in normals (12.9 mm). Proximal lesions were associated with much shorter left mainstems (9.1 mm) than distal ones (12.9 mm) ($P < 0.005$).

There was no difference in the mean angle of

bifurcation either between angiograms with (81°) and without (75°) stenoses ($P > 0.05$) or between proximal (85°) and distal (76°) disease groups ($P > 0.1$). In combination with mainstem length, a wide bifurcation angle exerted a slight ($0.05 < P < 0.1$) additional influence favouring proximal localisation.

Dominant circumflex artery systems possessed mean left main coronary arteries (9.1 mm) significantly shorter ($P < 0.05$) than right coronary artery dominant ones (12.0 mm) but both had identical disease distribution.

Short left main coronary arteries favour proximal localisation of stenoses. The angle of bifurcation alone exerted no influence but in combination with a short left main coronary artery a wide angle slightly enhanced proximal distribution. The poor prognosis of proximal lesions and their suitability for bypass grafting make these relations important.

Experimental investigation of arteriographic findings that suggest coronary spasm

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Experiments were undertaken in anaesthetised dogs to test the hypothesis that transient narrowing or disappearance of a coronary artery during arteriography may not necessarily be the result of coronary spasm. The left anterior descending coronary artery was narrowed in open and closed chest experiments. Atrial pacing (in 5) and ergonovine maleate, 0.4 mg (in 5) were then used to produce acute regional myocardial ischaemia. Simultaneous left ventricular and left coronary angiograms were recorded before and after stenosis of the left anterior descending artery and then during ischaemia. Intravenous glyceryl trinitrate (10 µg/ml per min) was then infused and the angiograms repeated. In a further 5 dogs an injection of microspheres into the left anterior descending artery alone was used to produce regional ischaemia. In all the dogs the left anterior descending artery was opacified throughout its length before and after stenosis with no evidence of regional ischaemia. During regional ischaemia the portion of the left anterior descending artery associated with the dyskinetic segment failed to opacify. This was reversed by using intravenous glyceryl trinitrate. The same experiments using stenosis of the left anterior descending artery and

atrial pacing were performed in 10 dogs while assessing changes in regional myocardial perfusion using krypton-81 m and a gamma camera. Atrial pacing produced a redistribution of perfusion with a 78 ± 14 per cent decrease in blood flow in the affected segment. This study suggests that haemodynamic and mechanical factors that affect coronary flow during acute regional ischaemia can cause the transient narrowing or disappearance of a coronary artery. The angiographer should consider that the arteriographic appearances of the coronary arteries may be temporarily changed during episodes of acute regional myocardial ischaemia with altered ventricular function.

Evidence for enhanced heart rate response to repeated treadmill testing uninfluenced by propranolol in stable angina

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Nine normotensive male patients with stable angina, no previous history of myocardial infarction, a normal chest x-ray film and resting electrocardiogram, but with a positive electrocardiographic response to exercise were studied. Each patient underwent Bruce procedure treadmill electrocardiography at 0800, 1200, and 1600 hours on one day, the end-point being angina or exhaustion. The maximal and recovery heart rates were significantly greater in the afternoon than in the morning ($P < 0.025$) and the ST segment changes were still significantly greater ($P < .002$) after 10 minutes recovery, in spite of comparable walking times. In 9 normal subjects there was no such diurnal variation in exercise response.

Nine further patients who had been receiving propranolol 40 mg qds for at least a month were studied in a similar way and received their medication after each treadmill trip. There was no significant difference in the mean age, weight, or serum cholesterol between the two groups. The treated group also had higher afternoon maximal and recovery heart rates which were significantly greater until 3 minutes into the recovery period ($P < 0.025$), but the ST segment changes were greater only until the end of the first minute ($P < 0.05$). When the treated group was retested the same effect was shown and the results were also valid tested diagonally across the interval which was 6 weeks. Possible mechanisms and the implications of these observations were discussed.

Biochemical effects of beta₂ adrenergic blockade in patients undergoing cardiopulmonary bypass

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In a previous retrospective study a paradoxical rise in serum potassium during cardiopulmonary bypass was observed in patients taking propranolol or oxprenolol. A prospective comparison of propranolol with the beta₁ adrenergic antagonist metoprolol was therefore undertaken in patients undergoing surgery for coronary artery disease. The 10 patients in each group were comparable with respect to age, duration of symptoms, etc, and both groups were managed according to a strict procedure. Propranolol or metoprolol was continued until surgery and no blood or potassium supplements were administered during the study. Mean serum potassium rose by 1.2 mmol/l during cardiopulmonary bypass in the propranolol group and fell by 0.08 mmol/l in the metoprolol group ($P \neq < 0.001$). Urinary potassium loss was similar in both groups. After an initial glucose load serum glucose level fell significantly in the propranolol group as compared with the metoprolol group.

Disturbances of potassium homeostasis after beta₂ blockade have been shown in animal experiments and our results indicate a relation between beta₂ blockade and hyperkalaemia during cardiopulmonary bypass; this may be associated with intracellular substrate deficiency and subsequent myocardial failure.

Potential protective value of enhanced myocardial carbohydrate utilisation with reduced free fatty acid uptake after UK 25842 in patients with coronary artery disease

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The influence of UK 25842 (L-4-hydroxyphenylglycine) in a dose of 4 to 12 mg/kg body weight on haemodynamics and myocardial metabolism was investigated in 20 patients with obstructive coronary artery disease before and during angina pectoris induced by atrial pacing. Heart rate, intracardiac pressures, cardiac output, and coronary venous blood flow were measured. Myocardial uptake and extraction of lactate, pyruvate, free

fatty acids, acetoacetate, glycerol, and triglycerides were determined from systemic arterial and coronary sinus blood samples.

No major haemodynamic changes or any side effects occurred after UK 25842. Atrial pacing time to the onset of angina increased from 211 ± 29 to 290 ± 27 seconds after 12 mg/kg UK 25842.

Both myocardial free fatty acid uptake and extraction ratios fell. During angina myocardial free fatty acid uptake was reduced from 31.5 ± 6.3 to 17.4 ± 4.8 $\mu\text{mol/min}$. ($P < 0.05$). There was a corresponding increase in myocardial uptake of lactate of -25.5 to $+14.4$ $\mu\text{mol/min}$ (-2.3 to $+1.3$ mg/min) and of pyruvate of 2.3 to 13.7 $\mu\text{mol/min}$ (0.2 to 1.2 mg/min), $P < 0.01$, with reversal of lactate production in 60 per cent of patients. Myocardial glucose uptake also increased. This change from free fatty acid to carbohydrate utilisation by the myocardium was confirmed by an increase in the myocardial RQ from 0.8 to 1.02. These changes were associated with a reduction in myocardial oxygen consumption (MVO_2) from 23.5 to 19.9 ml/min.

This reduction in MVO_2 associated with enhanced myocardial carbohydrate utilisation, produced by UK 25842, has great potential value not only in patients with angina but also in those with myocardial infarction, when jeopardised areas of the myocardium may be salvaged.

Cardiac emission tomography with thallium 201: new approach to perfusion scintigraphy

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This report describes the use of a new device for myocardial imaging in humans by emission computerised tomography. The dual-detector emission tomograph rotates in 6 degree increments, at each of which a 40 cm linear scan is performed. A total of 30 rotations complete a single scan, taking between 4 and 15 minutes. The slice image is reconstructed into an 80×80 matrix.

The tomograph has been used to image the distribution of thallium 201 in a group including normals, patients with left ventricular aneurysms, patients with myocardial infarction, and patients with chest pain. All patients have been studied both by conventional gamma camera imaging (4 projections), and by emission computerised tomo-

graphy (average of 3 transverse sections).

Patients with chest pain were injected after exercise and were studied both initially and after a 4-hour delay to permit redistribution. The remainder were studied only at rest. The initial results have indicated that exercise perfusion defects not seen on the conventional images can be unmasked by emission computerised tomography. The spatial separation between the liver and spleen and the heart on the image may yield superior results in the assessment of apical perfusion defects. The addition of depth information may lead to improved scintigraphic assessment of infarct size and allow detection of subendocardial ischaemic zones.

Pulmonary perfusion imaging applied to prediction of pulmonary vascular pressures

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The effect of gravity on pulmonary blood flow is well established. Normally the lung bases are better perfused than the upper zones but this difference disappears as left atrial pressure rises. Theoretically radionuclide lung imaging should reflect this change quantitatively but previous attempts to show this have met with mixed success. We have investigated 30 patients with valvar heart disease undergoing cardiac catheterisation and have used a new radionuclide method. Perfusion images were recorded in anterior and posterior views. On the computer, counts from the upper half (UZ) and the total (T) of the images were made and the geometric mean of the two views calculated to give composite UZ and T counts. The ratio UZ/T was plotted against mean pulmonary wedge pressure (MPWP) and mean pulmonary artery pressure (MPAP). Good correlation was found between UZ/T and MPWP ($r = 0.95$); correlation with MPAP was less satisfactory ($r = 0.72$). The data include those from 4 patients studied both before and after corrective surgery (with postoperative cardiac catheterisation) when the change in the UZ/T ratio reflected the reduction in MPWP achieved. This simply-obtained non-invasive measurement may be used as a reliable index of the height of MPWP diagnostically, and for sequential studies.

Quantitative thallium 201 myocardial imaging in coronary artery disease

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Myocardial imaging using thallium 201 (^{201}Tl) was performed in 60 subjects who achieved predicted maximal exercise performance without evidence of cardiovascular abnormality. Computer processing of the images was carried out to provide quantitative information on regional myocardial ^{201}Tl distribution, and 'normal' variation was determined.

This processing technique was applied to rest and exercise ^{201}Tl myocardial images in 170 patients with exertional chest pain who were subsequently shown to have anatomically significant coronary artery disease.

Exercise ^{201}Tl myocardial images suggestive of ischaemia were seen in 126 patients (74%) compared with 111 (65%) who developed chest pain during exercise and 94 (55%) with electrocardiographic evidence of ischaemia. Patients with a history of atypical chest pain had a lower incidence of 'ischaemia' (54%) than patients with classical angina pectoris (80%). The demonstration of defects on exercise ^{201}Tl images increased with the extent and severity of the underlying coronary artery disease but was uninfluenced by the presence of previous myocardial infarction. Abnormal exercise ^{201}Tl images were seen in 110 of 144 patients (76%) achieving maximal symptom-limited exercise but in only 16 of 37 patients (43%) exercised to sub-maximal levels.

Exercise ^{201}Tl myocardial imaging is more sensitive than other exercise indices in the detection and assessment of coronary artery disease, but this is dependent on achieving maximal symptom-limited exercise. Simple quantification aids the interpretation of these images.

Exercise first-pass radionuclide ventriculography in patients with coronary artery disease

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Thirty-four patients with stable angina pectoris and coronary artery disease were studied by computerised first-pass radionuclide ventriculography at exercise-induced angina using a multicrystal gamma camera. Ejection fraction fell significantly ($P < 0.0005$) from a mean of 61.1 rest to a mean of 52.9

on exercise. Extent of coronary artery disease was judged from coronary angiograms; a stenosis of greater than 50 per cent was regarded as significant. Ejection fraction fell significantly for the groups with triple vessel disease ($P < 0.0005$) and with less extensive lesions ($P < 0.05$). Fall in ejection fraction was significantly greater for those patients with triple vessel disease ($P < 0.01$). All 5 patients with a fall in ejection fraction of greater than 15 per cent had triple vessel disease. There was no significant change in ejection fraction in a control group

of normal patients. Wall motion abnormalities were assessed using a hemiaxial model. Thirty-four abnormal segments were induced by exercise; 10 anterior, 11 inferior, and 13 apical. All except 2 (1 inferior, 1 apical) were in regions supplied by significantly stenosed vessels. Exercise first-pass radionuclide ventriculography may be performed during angina to show changes in regional and general myocardial function. It is a useful screening test for coronary artery disease and may especially identify patients at high risk.